Fatal Epistaxis

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• **The instances in which nosebleed is potentially fatal are those in which there is a history of recent head injury, severe arteriosclerotic cardiovascular disease or an underlying vascular tumor in the nasal chambers. Fatal nasal bleeding has not been reported in children. An awareness on the part of the physician of the potentially fatal significance of his patient's nosebleed is the very best insurance against such an event. Intelligent history-taking, careful physical and x-ray examination, generous sedation, precise local cauterization and packing, estimation of hemoglobin mass and a search for bleeding and clotting disorders are the best weapons of the physician called to treat epistaxis. These procedures, coupled with adequate blood replacement and an informed attitude toward surgical interruption of the blood supply to the bleeding region should forestall death from fatal nosebleed.**

ON THE NIGHT of November 27, 1957, a 27-year-old Negro man was admitted to Wadsworth General Hospital, Veterans Administration Center, Los Angeles, with a diagnosis of anemia of undetermined origin. Late that night, the genesis of the anemia became clear when a massive nasal hemorrhage developed that responded only to the insertion of a large postnasal pack. Questioning of the patient and his relatives elicited that three months before he had been in hospital elsewhere following a motorcycle accident in which he received a closed head injury that resulted in left hemiparesis and recurrent nosebleeds. The patient said that he had not had nosebleeds before the accident and that the episodes of bleeding were not preceded by nasal manipulation or physical exertion. There was no history of sickle cell disease, easy bruising, jaundice or familial bleeding tendencies.

The patient was pale and his memory poor. Also noted were left peripheral facial nerve paralysis, diminished sensation over the distribution of the left trigeminal nerve and a conductive hearing loss in the left ear. The hematocrit was 24 per cent, the hemoglobin content was 7.2 gm. per 100 cc. of blood, leukocytes numbered 5,050 per cu. mm. with a cell differential within normal limits, and bleeding and clotting times were normal. A Rumpel-Leede test was negative for capillary fragility. Careful review of the roentgenograms of the skull and face showed only a fracture of the left zygoma.

During his stay in the hospital the patient had several episodes of sudden, spontaneous, massive bleeding from the nose and mouth, controlled only by insertion of large postnasal packs and necessitating several transfusions. Because of the persistence and severity of these nosebleeds, the left external carotid artery was ligated on the morning of December 14, 1957. Nasal packing was removed and the patient apparently did well following this operation until, at 6 p.m., cataclysmic hemorrhage from the nasopharynx developed suddenly. Within three minutes of the onset of this hemorrhage a postnasal pack and anterior nasal pack were reinserted and a venous cutdown was begun. In five minutes, however, the patient was dead.

At autopsy a rent was noted in the intracranial portion of the left internal carotid artery which communicated with the interior of the sphenoid sinus and through the sphenoid ostium with the nasopharynx. Death might possibly have been averted had carotid angiography been included in the diagnostic studies which were undertaken to find the cause of epistaxis.

CASE 2. On December 30, 1957, a 68-year-old white man entered Wadsworth General Hospital for treatment of severe left-sided epistaxis. In January of 1956 he had been in hospital for the same disorder and, at that time, was bleeding from a point below the posterior tip of the left inferior turbinate. In April of 1957, he had been brought into the hospital for treatment of arteriosclerotic heart disease with angina pectoris. A recent examination directed at a disturbance of bowel function had indicated the presence of an annular lesion of the large bowel. On December 29, 1957, bleeding again commenced from the left side of the nose and the patient was brought into the hospital on the following day. There was no other history of bleeding diathesis.

Upon physical examination he was observed to be pale, calm and fairly alert, with definite slurring of speech but no obvious motor weakness. He complained of continuous pain in the chest and in both

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arms. The blood pressure was 150/90 mm. of mercury. The pulse rate and temperature were within normal limits. Upon removal of a nasal pack that had been placed to stop the bleeding, epistaxis did not recur. No evidence was seen of tumor or infection of the nasal chambers or paranasal sinuses. The heart was not enlarged and there were no murmurs or rubs. The lungs were clear. Hemoglobin was 9.8 gm. per 100 cc. of blood and the hematocrit was 29 per cent. Leukocytes numbered 5,500 per cu. mm. A roentgenogram of the chest showed the heart, great vessels and lung fields to be within normal limits. An electrocardiogram was interpreted as consistent with subendocardial injury.

The patient was treated with nitroglycerin, ferrous sulfate, sedatives and bed rest. Bleeding from the nose did not recur, but the patient continued to complain of discomfort in the chest and arms. Because of the anemia and of coronary insufficiency indicated by progressive electrocardiographic changes, a transfusion was begun at 6:30 p.m. on January 7, 1958. Within a half hour the patient began to perspire profusely and the skin became ashen. He complained of fairly severe pain in the chest. At 8:15 p.m. respirations became labored and oxygen was administered. At 8:30 p.m. the patient died.

Permission to perform examination postmortem could not be obtained. While the nosebleed in that case may appear to have been incidental to the already existing coronary artery disease and bowel carcinoma, the addition of blood loss may have upset the tenuous cardiovascular equilibrium enough to bring about intolerable coronary insufficiency. The possibility that death was caused by a reaction to the transfusion was considered, but was discarded after consultation with the medical staff and examination of the blood remaining in the transfusion flask for mismatching and bacterial contamination.

The feelings of profound chagrin and defeat arising from my personal experience in treating these two patients led me to search the literature for instances in which epistaxis, generally a banal and simply bothersome affliction, had assumed fatal proportions. The search was well rewarded. It turned out that the two cases described above were illustrative examples of the pathologic events culminating in death from nosebleed: A review of the records of the U. S. Veterans Administration Hospital at Los Angeles from 1949 to 1958 disclosed two additional cases of fatal epistaxis.

Nosebleeds spare no age group. They occur in the toddler who falls and strikes his nose, in the nervous nose-picker ("epistaxis digitorum") and in elderly arteriosclerotic persons. The usual local causes are foreign body, instrumental or digital manipulation, blows to the nose and infections and tumors of the nasal chambers. The generalized conditions which can lead to nasal hemorrhage are blood disturbances, such as leukemia and polycythemia, and disorders of the coagulation mechanism, as well as rheumatic fever and scurvy. Increased venous pressure as from emphysema, prolonged coughing or straining and tumors of the neck and upper mediastinum have also been cited as causative conditions. The nosebleeds that physicians most often see in children and adults are those associated with blunt trauma to the nose or maladroit cleansing of the nasal passages. Nosebleeds among the elderly are distressingly frequent, usually arising from the posterior portion of the nasal fossa and generally defying etiologic diagnosis. In the older patients, epistaxis is often associated with increased arterial pressure and evidences of arteriosclerosis. The association may be fortuitous, however, for hypertension and medical sclerosis are recognized manifestations of the senium, and nosebleed is commonly seen in their absence.

Halberg[7] effectively summarized the etiology of epistaxis in his report of 212 cases at the Mayo Clinic. In that series, 50.9 per cent of patients were over 54 years of age and in approximately 80 per cent of them the bleeding ceased spontaneously.

In his diagnostic endeavors the clinician is limited to a search for the most common causative factors and must attempt to exclude potentially serious but undiscovered illness of which the nasal hemorrhage may be the first sign. When considering fatal epistaxis, we must pay particular attention to the nasal hemorrhage that follows a head injury. Here, the basilar skull fracture has a definite role in that the line of fracture may intersect the course of a major blood vessel with resulting hemorrhage into the nasal chambers, paranasal sinuses, eustachian tube or nasopharynx. Additionally, these fractures of the floor of the brain case are difficult to detect radiologically, often remaining undiagnosed until postmortem examination. As a general rule, the injuries that result in the fortunately rare instances of massive, life-threatening epistaxis are those in which the anterior ethmoid artery or the internal carotid artery is torn. Injury to the latter vessel is sometimes associated with disturbances of vision, of facial sensation or of pituitary function and is accompanied by premonitory aura or intracranial bruit.

REVIEW OF THE LITERATURE

In a review of the medical literature for the preceding 100 years, reports were found of a number of cases of fatal nosebleed. The first account of such a case was given by Levy[10] who, in 1896, described a 20-year-old white male miner who was struck on

the jaw in an occupational accident in Colorado. Immediately after the injury severe epistaxis developed. It recurred repeatedly and was associated with suppurative otitis media and mastoiditis. Thirty-eight days after the injury, loss of blood from a nosebleed was great enough to cause death. Autopsy showed a "septic fistula" between the middle meningeal artery and the eustachian tube in the region of the foramen spinosum.

Matthews[11] in 1898 briefly mentioned a case of spontaneous fatal nasal hemorrhage in a 19-year-old boy who had "bilious fever." In 1899 Dock[6] discussed a 37-year-old man who had a fatal nasal hemorrhage arising from an "endothelioma carcinomatosum" of the turbinated nasal bones.

Kummel[9] in 1928 recounted the case of a 23-year-old man who received a head injury in a motorcycle accident. The blow left him unconscious and bleeding from the nose, mouth and right ear. He recovered rapidly but thereafter had spontaneous, severe epistaxis at approximately two-week intervals, accompanied by severe temporal headache which was worse on the left side. Sixty-eight days after the injury, total amaurosis supervened in the right eye, which showed a pale disc and extremely narrow retinal vessels. Ten weeks after the accident, the patient suddenly had tremendous hemorrhage from the nose and mouth and died within several minutes. Autopsy indicated that the bleeding was a result of a fissure in the thin bony wall between the sphenoid sinus of the internal carotid artery which communicated with a small laceration of the artery itself. This situation had led to the formation of an aneurysm of this vessel which produced pressure upon the second branch of the trigeminal, the sphenopalatine and, presumably, the optic nerves, resulting in the temporal headaches and blindness.

Bonnet,[3] in 1935, presented a similar instance of traumatic aneurysm of the internal carotid artery within the sphenoid sinus in a 31-year-old woman who three months earlier had had a fracture of the floor of the anterior fossa from a blow on the frontal region. Epistaxis which occurred immediately following the injury was treated by ligation of the external carotid artery, without success. Within three months of the injury, the right eye had become blind and the oculomotor nerve on that side had become paralyzed. Angiograms showed an intrasphenoidal carotid aneurysm. Ligation of the right internal carotid artery did not prevent a fatal nasal hemorrhage.

Bean,[1] in 1937, in a study of conditions which predisposed to myocardial infarction, cited one case in which severe epistaxis had led to a profound anemia and death. At postmortem examination, a fresh myocardial infarction was found. He also reported a case of fatal myocardial infarction following hemorrhage from a peptic ulcer, as well as two cases of infarction in patients who had profound anemia.

In 1939, Davis[5] recorded the case of a 17-year-old boy who was struck in the nose by a fist and received fractures of the nasal bones and the nasal processes of the maxilla. This was attended by severe epistaxis which eluded all attempts at control short of ligation of the major vessels and caused death on the night of injury. Postmortem examination showed the anterior ethmoid artery to be ruptured.

Rasquin,[13] in 1949, described his experience with a 46-year-old farmer who, for two years, endured recurrent epistaxis preceded by an aura of painful pulsations and a "heavy feeling" in the left temporal region. The nosebleeds were as remarkable for their brevity as for their severity. At one point the patient lost 950 cc. of blood in just 20 seconds. Exploration of the left maxillary sinus, ligation of the internal maxillary artery in the pterygomaxillary fossa, ligation of the external carotid artery and the application of intranasal radium therapy did not halt the episodes of nasal hemorrhage. As the bleeding continued, hemiplegia developed and the patient died two days later. Postmortem examination was not performed, but the author suggested the possibility of an aneurysm of the internal carotid artery as the underlying cause of the hemorrhages.

Bourdon,[4] in 1952, reported the case of a 30-year-old man with spontaneous persistent epistaxis despite the appearance of excellent general health. Within a few weeks fever and leukocytosis developed; the peripheral blood assumed the appearance of leukemia. Seven days later the epistaxis ceased, but on the following day the patient died. Postmortem examination was not reported.

Sigiuer,[15] in 1954, told of a 50-year-old white man who had the florid complexion typical of alcoholics and hepatosplenomegaly suggestive of Laennec's cirrhosis. The patient steadfastly denied the use of alcohol in any form or quantity, stating that his father had died a drunkard and as a result he himself had formed a lasting antipathy to liquor. The patient had been plagued with nosebleeds since the age of six years. Further, his father, brothers and two nephews had the same affliction. One brother died of spontaneous hemothorax, secondary to a pulmonary arteriovenous aneurysm. Upon careful examination of the patient, multiple, mucosal telangiectases were observed in the oral and nasal cavities. Despite vigorous local treatment, the patient died of a spontaneous nasal hemorrhage, a consequence of Osler-Weber-Rendu syndrome.

Hrenoff,[8] in 1954, wrote of his experiences in the treatment of a 61-year-old hypertensive man who had severe recurrent epistaxis that reduced the hemoglobin content to 7 gm. per 100 cc. of blood. The

patient was given Bistrium® (hexamethonium) to reduce the arterial pressure, but the patient died that night. The Bistrium® combined with the anemia due to blood loss caused fatal circulatory collapse.

Seftel,[14] in 1959, discussed epistaxis resulting from rupture of an intracranial aneurysm and presented the case of a 20-year-old man who died of exsanguination from this cause five months after receiving a head injury. He had had repeated, small nosebleeds in the interim and the fatal hemorrhage was from the nose and mouth.

From these recorded experiences, we see that death from nasal hemorrhage may be the result of simple exsanguination or it can represent the combination of a preexisting limited circulatory reserve upon which profound anemia is suddenly imposed. Loss of blood great enough to cause death may follow a blow to the head or may be associated with defective blood coagulation, with nasal hemangiomata or with idiopathic aneurysm of the internal carotid artery.

DIAGNOSTIC PROCEDURES

A careful history is always desirable but not always obtainable when dealing with a person whose overwhelming concern is the arrest of nasal hemorrhage. Once the bleeding has been controlled, it is of importance to search the patient's history for possible clues as to the genesis. Questions should be asked that will bring to light any diseases of the cardiovascular system, the blood-forming tissues and the blood-coagulating mechanism. The presence of a neoplasm in the nose or paranasal sinuses must be suspected, and the patient should be questioned carefully with regard to recent head injury and nasal trauma. The interrogator is wise also to pay close attention to symptoms suggesting coronary artery insufficiency or cerebrovascular disease, for these conditions can assume critical importance in the presence of sudden severe blood loss.

Of utmost importance in the definitive treatment of severe epistaxis by cautery, packing or ligation is the accurate localization of the source of the bleeding. For this purpose, a neurosurgical suction tip supported by a vigorous suction machine, an optically accurate head-mirror and a strong source of illumination are desirable. The importance of accurate identification of the bleeding area cannot be overstressed, particularly in cases in which ligation of the major vessel appears necessary; for the decision concerning which vessel to ligate rests largely upon this observation. As a matter of routine, determinations should be made of the hemoglobin mass, of bleeding and clotting time and prothrombin time and the concentration of platelets. Morphologic studies of the blood should be carried out.

THERAPY

The most desirable methods of arresting nasal bleeding are those that compress or destroy the bleeding vessel yet inflict a minimum of injury to the surrounding structures and cause the least discomfort to the patient. Precise, firm packing over the offending vessels, either alone or in combination with electrical or chemical coagulation of the bleeding point, will control nosebleed in most cases. The details of various methods of local therapy have been splendidly presented by Ogura[12] and Beinfield.[2]

In general the most troublesome nasal hemorrhages arise from the relatively inaccessible regions of the olfactory cleft and the posterior choanae. In these cases cauterization is often impossible and bleeding cannot be controlled by anterior packing. In such circumstances a posterior choanal or nasopharyngeal pack may be required, not once, but repeatedly. Various forms of systemic therapy, including administration of vitamins C, K, and P, together with estrogenic substances and pituitrins, have been recommended.

While centering attention on the source of bleeding, the physician must be mindful of the effect of blood loss upon the patient, watchful for signs of approaching hypovolemic shock and ready to order intravenous infusion of plasma expanders or whole blood. It is not the presence of blood in the nose but the lack of blood in the vascular system that causes death.

Generous sedation optimally in the form of morphine sulfate is both the gesture of kindness and an effective hemostatic maneuver. Anxious, tense patients bleed more freely, probably as a result of the elevated arterial pressure in combination with the nasal vascular engorgement seen in emotional stress. Frequently overhearing a telephoned order for morphine sulfate will stop the bleeding before the physician arrives on the ward.

Appropriate surgical treatment of severe nosebleed includes a number of techniques. One is sometimes required to reduce a severely deflected nasal septum that is hiding the source of the bleeding. Should the bleeding point lie upon the septum itself, the scarring that follows the operation will often permanently obliterate the vessel. In cases of posterior epistaxis, it is sometimes expedient to infract the inferior turbinate on the side of the bleeding, thus obtaining clear access to the posterior portion to the inferior nasal meatus, the most common site of posterior nasal hemorrhage. Until recently ligation of major arteries for the control of epistaxis had been regarded as a last resort, to be used only when the hemorrhage assumed life-threatening proportions. The earliest ligations for this purpose were

performed upon the common carotid artery. The mortality from this procedure relegated it to the hands of a surgeon "at bay." Today, the external carotid artery and the anterior ethmoid artery are the vessels most commonly interrupted in the treatment of severe nasal bleeding. The techniques of ligation employed upon these vessels carry with them a very small morbidity and no mortality whatsoever, as far as has been reported. The operations can be performed under local anesthesia and leave a minimum of external scarring. When the point of origin of the nosebleed can be accurately localized to the areas subserved by either of these vessels, arterial ligation is followed by prompt and permanent cessation of epistaxis.

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